Appendix M
Health and Safety

M.1 INTRODUCTION

This appendix presents detailed information on the potential impacts and risks to humans associated with releases of radioactivity and hazardous chemicals from the proposed storage and disposition technologies during normal operations and from postulated accidents. This information is intended to support the public and occupational health and safety assessments described in Sections 4.2 and 4.3 of this programmatic environmental impact statement (PEIS). Section M.2 provides information on normal radiological impacts, Section M.3 provides information on normal hazardous chemical impacts, Section M.4 provides information on human health and epidemiologic studies, and Section M.5 provides information on postulated facility accidents.

M.2 RADIOLOGICAL IMPACTS TO HUMAN HEALTH DURING NORMAL OPERATIONS

This section presents supporting information on the potential radiological impacts of normal operation to humans. This section provides the reader with background information on the nature of radiation (Section M.2.1), the methodology used to calculate radiological impacts (Section M.2.2), radiological releases from fissile material storage and disposition facilities (Section M.2.3), and radiological impacts from various fissile material storage and disposition facilities at each site (Sections M.2.4 through M.2.15).

A further description of the methodology used to assess the normal radiological impacts presented in this appendix and a detailed listing of the data used in the assessments are given in *Health Risk Data for Storage and Disposition of Weapons-Usable Fissile Materials* (Health Risk Data, October 1996).

M.2.1 BACKGROUND

M.2.1.1 Nature of Radiation and Its Effects on Humans

What is Radiation? Humans are constantly exposed to radiation from the solar system and from the earth's rocks and soil. This radiation contributes to the natural background radiation x ray that has always been around us. But there are also manmade sources of radiation, such as medical and dental x rays, household smoke detectors, and materials released from nuclear and coal-fired powerplants.

All matter in the universe is composed of atoms, and radiation comes from the activity of these tiny particles. Atoms are made up of even smaller particles (protons, neutrons, electrons). The number and arrangement of these particles distinguishes one atom from another.

Atoms of different types are known as elements. There are over 100 natural and manmade elements. Some of these elements, such as uranium (U), radium, plutonium (Pu), and thorium, share a very important quality: they are unstable. As they change into more stable forms, invisible waves of energy or particles, known as ionizing radiation, are released. Radioactivity is the emitting of this radiation.

Ionizing radiation refers to the fact that this energy force can ionize, or electrically charge atoms by stripping off electrons. Ionizing radiation can cause a change in the chemical composition of many things, including living tissue (organs), which can affect the way they function.

The effects on people of radiation that is emitted during disintegration (decay) of a radioactive substance depends on the kind of radiation (alpha and beta particles, and gamma and x rays) and the total amount of

radiation energy absorbed by the body. The total energy absorbed per unit quantity of tissue is referred to as absorbed dose. The absorbed dose, when multiplied by certain quality factors and factors that take into account different sensitivities of various tissues, is referred to as effective dose equivalent, or where the context is clear, simply dose. The common unit of effective dose equivalent is the roentgen equivalent man (rem) (1 rem equals 1,000 millirem [mrem]).

Alpha particles are the heaviest of these direct types of ionizing radiation, and despite a speed of about 16,000 kilometers (km)/second (s) (9,940 miles [mi]/s), they can travel only several centimeters in air. Alpha particles lose their energy almost as soon as they collide with anything. They can easily be stopped by a sheet of paper or the skin's surface.

Beta particles are much lighter than alpha particles. They can travel as much as 160,000 km/s (99,400 mi/s) and can travel in the air for a distance of about 3 meters (m) (9.8 feet [ft]). Beta particles can pass through a sheet of paper, but may be stopped by a thin sheet of aluminum foil or glass.

Gamma and x rays, unlike alpha or beta particles, are waves of pure energy. Gamma rays travel at the speed of light (300,000 km/s [186,000 mi/s]). Gamma radiation is very penetrating and requires a thick wall of concrete, lead, or steel to stop it.

The neutron is another particle which contributes to radiation exposure, both directly and indirectly. The latter is associated with the gamma rays and alpha particles which are emitted following neutron capture in matter. A neutron has about one quarter the weight of an alpha particle and can travel at speeds of up to 39,000 km/s (24,200 mi/s). Neutrons are more penetrating than beta particles, but less than gamma rays.

The radioactivity of a material decreases with time. The time it takes a material to lose half of its original radioactivity is its half-life. For example, a quantity of iodine-131, a material that has a half-life of 8 days, will lose one-half of its radioactivity in that amount of time. In 8 more days, one-half of the remaining radioactivity will be lost, and so on. Eventually, the radioactivity will essentially disappear. Each radioactive element has a characteristic half-life. The half-lives of various radioactive elements may vary from millionths of a second to millions of years.

As a radioactive element gives up its radioactivity, it often changes to an entirely different element, one that may or may not be radioactive. Eventually, a stable element is formed. This transformation may take place in several steps and is known as a decay chain. Radium, for example, is a naturally occurring radioactive element with a half-life of 1,622 years. It emits an alpha particle and becomes radon, a radioactive gas with a half-life of only 3.8 days. Radon decays to polonium and through a series of steps to bismuth and ultimately to lead.

Units of Radiation Measure. Scientists and engineers use a variety of units to measure radiation. These different units can be used to determine the amount, type and intensity of radiation. Just as heat can be measured in terms of its intensity or its effects using units of calories or degrees, amount of radiation can be measured in curies (Ci), radiation absorbed doses (rads), or rems.

The curie, named after the French scientists Marie and Pierre Curie, describes the "intensity" of a sample of radioactive material. The rate of decay of 1 gram of radium is the basis of this unit of measure. It is equal to 3.7×10^{10} disintegrations (decays)/s.

The total energy absorbed per unit quantity of tissue is referred to as absorbed dose. The rad is the unit of measurement for the physical absorption of radiation. Much like sunlight heats the pavement by giving up an amount of energy to it, radiation gives up rads of energy to objects in its path. One rad is equal to the amount of radiation that leads to the deposition of 0.01 joule of energy per kilogram of absorbing material.

A rem is a measurement of the dose from radiation based on its biological effects. The rem is used to measure the effects of radiation on the body, much like degrees Centigrade can be used to measure the effects of sunlight heating pavement. Thus, 1 rem of one type of radiation is presumed to have the same biological effects as 1 rem of any other kind of radiation. This standard allows comparison of the biological effects of radionuclides that emit different types of radiation.

An individual may be exposed to ionizing radiation externally from a radioactive source outside the body, and/or internally from ingesting radioactive material. The external dose is different from the internal dose. An external dose is delivered only during the actual time of exposure to the external radiation source. An internal dose, however, continues to be delivered as long as the radioactive source is in the body, although both radioactive decay and elimination of the radionuclide by ordinary metabolic processes decrease the dose rate with the passage of time. The dose from internal exposure is calculated over 50 years following the initial exposure.

The three types of doses calculated in this include an external dose, an internal dose, and a combined external and internal dose. Each type of dose is discussed separately below.

External Dose. The external dose can arise from several different pathways. All these pathways have in common the fact that the radiation causing the exposure is external to the body. In this PEIS, these pathways include exposure to a cloud of radiation passing overhead of the receptor, standing on ground which is contaminated with radioactivity, swimming in contaminated water, and boating in contaminated water. The appropriate measure of dose is called the effective dose equivalent. It should be noted that if the receptor departs from the source of radiation exposure, his dose rate will be reduced. It is assumed that external exposure occurs uniformly during the year.

Internal Dose. The internal dose arises from a radiation source entering the human body through either ingestion of contaminated food and water or inhalation of contaminated air. In this PEIS, pathways for internal exposure include ingestion of crops contaminated either by airborne radiation depositing on the crops or by irrigation of crops using contaminated water sources, ingestion of animal products from animals that ingested contaminated food, ingestion of contaminated water, inhalation of contaminated air, and absorption of contaminated water through the skin during swimming. Unlike external exposures, once the radiation enters the body, it remains there for various periods of time that depend on decay and biological elimination rates. The unit of measure for internal doses is the committed dose equivalent. It is the internal dose that each body organ receives from 1 "year intake" (ingestion plus inhalation). Normally, a 50- or 70-year dose-commitment period is used (i.e., the 1 year intake period plus 49 or 69 years). The dose rate increases during the 1 year of intake. The dose rate, after the 1 year of intake, slowly declines as the radioactivity in the body continues to produce a dose. The integral of the dose rate over the 50 or 70 years gives the committed dose equivalent. In this PEIS, a 50-year, dose-commitment period was used.

The various organs of the body have different susceptibilities to harm from radiation. The quantity that takes these different susceptibilities into account to provide a broad indicator of the risk to the health of an individual from radiation is called the committed effective dose equivalent. It is obtained by multiplying the committed dose equivalent in each major organ or tissue by a weighting factor associated with the risk susceptibility of the tissue or organ, then summing the totals. It is possible that the committed dose equivalent to an organ is larger than the committed effective dose equivalent if that organ has a small weighting factor. The concept of committed effective dose equivalent applies only to internal pathways.

Combined External and Internal Dose. For convenience, the sum of the committed effective dose equivalent from internal pathways and the effective dose equivalent from external pathways is also called the committed effective dose equivalent in this PEIS (note that in Department of Energy [DOE] Order 5400.1, this quantity is called the effective dose equivalent).

The units used in this PEIS for committed dose equivalent, effective dose equivalent, and committed effective dose equivalent to an individual are the rem and mrem (1/1000 of 1 rem). The corresponding unit for the collective dose to a population (the sum of the doses to members of the population, or the product of the number of exposed individuals and their average dose) is the person-rem.

Sources of Radiation. The average American receives a total of about 350 mrem/year (yr) from all sources of radiation, both natural and manmade. The sources of radiation can be divided into six different categories: cosmic radiation, terrestrial radiation, internal radiation, consumer products, medical diagnosis and therapy, and other sources (NCRP 1987a:9-15). Each category is discussed below.

Cosmic radiation is ionizing radiation resulting from energetic charged particles from space continuously hitting the earth's atmosphere. These particles and the secondary particles and photons they create are cosmic radiation. Because the atmosphere provides some shielding against cosmic radiation, the intensity of this radiation increases with altitude above sea level. For the sites considered in this PEIS, the cosmic radiation ranged from 27 to 50 mrem/yr. The average dose to the people in the United States is about 27 mrem/yr.

External terrestrial radiation is the radiation emitted from the radioactive materials in the earth's rocks and soils. The average dose from external terrestrial radiation is about 28 mrem/yr. The external terrestrial radiation for the sites in this PEIS ranged from 15 to 63 mrem/yr.

Internal radiation arises from the human body metabolizing natural radioactive material which has entered the body by inhalation or ingestion. Natural radionuclides in the body include isotopes of U, thorium, radium, radon, polonium, bismuth, potassium, rubidium, and carbon. The major contributor to the annual dose equivalent for internal radioactivity are the short-lived decay products of radon which contribute about 200 mrem/yr. The average dose from other internal radionuclides is about 39 mrem/yr.

Consumer products also contain sources of ionizing radiation. In some products, like smoke detectors and airport x ray machines, the radiation source is essential to the products' operation. In other products, such as television and tobacco, the radiation occurs incidentally to the product function. The average dose is about 10 mrem/yr.

Radiation is an important diagnostic medical tool and cancer treatment. Diagnostic x rays result in an average exposure of 39 mrem/yr. Nuclear medical procedures result in an average exposure of 14 mrem/yr.

There are a few additional sources of radiation that contribute minor doses to individuals in the United States. The dose from nuclear fuel cycle facilities such as uranium mines, mills and fuel processing plants, nuclear power plants and transportation routes has been estimated to be less than 1 mrem per year. Radioactive fallout from atmospheric atomic bomb tests, emissions of radioactive material from DOE and Nuclear Regulatory Commission (NRC) licensed facilities, emissions from certain mineral extraction facilities, and transportation of radioactive materials contributes less than 1 mrem/yr to the average dose to an individual. Air travel contributes approximately 1 mrem/yr to the average dose.

The collective (or population) dose to an exposed population is calculated by summing the estimated doses received by each member of the exposed population. This total dose received by the exposed population is measured in person-rem. For example, if 1,000 people each received a dose of 1 mrem (0.001 rem), the collective dose is 1,000 persons x 0.001 rem = 1.0 person-rem. Alternatively, the same collective dose (1.0 person-rem) results from 500 people each of whom received a dose of 2 mrem (500 persons x 2 mrem = 1 person-rem).

Limits of Radiation Exposure. The amount of manmade radiation that the public may be exposed to is limited by Federal regulations. Although most scientists believe that radiation absorbed in small doses over several years is not harmful, U.S. Government regulations assume that the effects of all radiation exposures are cumulative.

Under the *Clean Air Act*, the exposure to a member of the general public from DOE facility releases into the atmosphere is limited by the Environmental Protection Agency (EPA) to a dose of 10 mrem/yr in addition to the natural background and medical radiation normally received (40 Code of Federal Regulations [CFR] 61, Subpart H). DOE also limits to 10 mrem the dose annually received from material released to the atmosphere (DOE Order 5400.5). The EPA and DOE also limit the annual dose to a member of the general public from radioactive releases to drinking water to 4 mrem, as required under the *Safe Drinking Water Act* (40 CFR 141; DOE Order 5400.5). The annual dose from all radiation sources from a site is limited by the EPA to 25 mrem (40 CFR 190). The DOE annual limit of radiation dose to a member of the general public from all DOE facilities is 100 mrem total, from all pathways (DOE Order 5400.5).

All DOE facilities covered by this PEIS operate well below this limit. It is estimated that the average individual in the United States receives a dose of about 0.3 rem (300 mrem) per year from natural sources of radiation. For perspective, a modern chest x ray results in an approximate dose of 0.006 rem (6 mrem), while a diagnostic pelvis and hip x ray results in an approximate dose of 0.065 rem (65 mrem) (NCRP 1987a:45). A person must receive an acute (short-term) dose of approximately 600 rem (600,000 mrem) before there is a high probability of near-term death (NAS 1990a:176).

For people working in an occupation that involves radiation, DOE and the NRC limit doses to 5 rem (5,000 mrem) in any 1 year (10 CFR 20; 10 CFR 835). For NRC-licensed facilities, the applicable site dose limits depend on the facility type. For other-than-power-reactors, the EPA limits discussed above apply. For power reactors, the annual total dose limit from all releases combined is the same as the EPA limit of 25 mrem (40 CFR 190). However, to demonstrate compliance with the as low as reasonably achievable philosophy, efforts must be made to further reduce releases to the guideline values given in Appendix I to 10 CFR 50.

M.2.1.2 Health Effects

Radiation exposure and its consequences are topics of interest to the general public. For this reason, this PEIS places much emphasis on the consequences of exposure to radiation, even though the effects of radiation exposure under most circumstances evaluated in this PEIS are small. This section explains the basic concepts used in the evaluation of radiation effects in order to provide the background for later discussion of impacts.

Radiation can cause a variety of ill-health effects in people. The most significant ill-health effect to depict the consequences of environmental and occupational radiation exposure is induction of cancer fatalities. This effect is referred to as "latent" cancer fatalities because the cancer may take many years to develop and for death to occur and may not actually be the cause of death. In the discussions which follow, it should be noted that all fatal cancers are latent and the term "latent" is not used.

Health impacts from radiation exposure, whether from sources external or internal to the body, generally are identified as "somatic" (affecting the individual exposed) or "genetic" (affecting descendants of the exposed individual). Radiation is more likely to produce somatic effects rather than genetic effects. Therefore, for this PEIS, only the somatic risks are presented. The somatic risks of most importance are the induction of cancers. Except for leukemia, which can have an induction period (time between exposure to carcinogen and cancer diagnosis) of as little as 2 to 7 years, most cancers have an induction period of more than 20 years.

For a uniform irradiation of the body, the incidence of cancer varies among organs and tissues; the thyroid and skin demonstrate a greater sensitivity than other organs. However, such cancers also produce relatively low mortality rates because they are relatively amenable to medical treatment. Because of the readily available data for cancer mortality rates and the relative scarcity of prospective epidemiologic studies, somatic effects leading to cancer fatalities rather than cancer incidence are presented in this PEIS. The numbers of cancer fatalities can be used to compare the risks among the various alternatives.

The fatal cancer risk estimators presented in this appendix for radiation technically apply only to low-linear energy transfer radiation (gamma rays and beta particles). However, on a per rem rather than a per rad basis, the fatal risk estimators are higher for this type of radiation than for high-linear energy transfer radiation (alpha particles). In this PEIS, the low-linear energy transfer risk estimators are conservatively assumed to apply to all radiation exposures.

The National Research Council's Committee on the Biological Effects of Ionizing Radiations (BEIR) has prepared a series of reports to advise the U.S. Government on the health consequences of radiation exposures. The latest of these reports, *Health Effects of Exposure to Low Levels of Ionizing Radiation BEIR V*, published in 1990, provides the most current estimates for excess mortality from leukemia and cancers other than leukemia expected to result from exposure to ionizing radiation. The BEIR V report updates the models and risk estimates provided in the earlier report of the BEIR III Committee, *The Effects of Populations of Exposure to Low-Levels of Ionizing Radiation,* published in 1980. The BEIR V models were developed for application to the U.S. population.

The BEIR V provides estimates that are consistently higher than those in BEIR III. This is attributed to several factors including the use of a linear dose response model for cancers other than leukemia, revised dosimetry for the Japanese atomic bomb survivors, and additional follow-up studies of the atomic bomb survivors and other cohorts. The BEIR III employs constant relative and absolute risk models, with separate coefficients for each of several sex and age-at-exposure groups, while BEIR V develops models in which the excess relative risk is expressed as a function of age at exposure, time after exposure, and sex for each of several cancer categories. The BEIR III models were based on the assumption that absolute risks are comparable between the atomic bomb survivors and the U.S. population, while BEIR V models were based on the assumption that the relative risks are comparable. For a disease such as lung cancer, where baseline risks in the United States are much larger than those in Japan, the BEIR V approach leads to larger risk estimates than the BEIR III approach.

The models and risk coefficients in BEIR V were derived through analyses of relevant epidemiologic data including the Japanese atomic bomb survivors, ankylosis spondylitis patients, Canadian and Massachusetts fluoroscopy patients (breast cancer), New York postpartum mastitis patients (breast cancer), Israel Tinea Capitis patients (thyroid cancer), and Rochester thymus patients (thyroid cancer). Models for leukemia, respiratory cancer, digestive cancer, and other cancers used only the atomic bomb survivor data, although results of analyses of the ankylosis spondylitis patients were considered. Atomic bomb survivor analyses were based on revised dosimetry with an assumed Relative Biological Effectiveness of 20 for neutrons, and were restricted to doses less than 400 rads. Estimates of risks of fatal cancers other than leukemia were obtained by totaling the estimates for breast cancer, respiratory cancer, digestive cancer and other cancers.

Risk Estimates for Doses Received During an Accident. The BEIR V includes risk estimates for a single exposure of 10 rem to a population of 100,000 people (1.0×10^6 person-rem). In this case, fatality estimates for leukemia, breast cancer, respiratory cancer, digestive cancer, and other cancers are given for both sexes and nine age-at-exposure groups. These estimates, based on the linear model, are summarized in Table M.2.1.2–1. The average risk estimate from all ages and both sexes is 885 excess cancer fatalities per million person-rem. This value has been conservatively rounded up to 1,000 excess cancer fatalities per million person-rem. Section M.5.1.3.2 contains additional discussions on accident risk estimators.

Although values for other health effects are not presented in this PEIS, the risk estimators for non-fatal cancers and for genetic disorders to future generations are estimated to be approximately 200 and 260 per million person-rem, respectively. These values are based on information presented in the *1990 Recommendations of the International Commission on Radiological Protection* (ICRP Publication 60) and are seen to be 20 percent and 26 percent, respectively, of the fatal cancer estimator. Thus, for example, if the number of excess fatal cancers is projected to be "X," the number of excess genetic disorders would be 0.26 times "X."

Risk Estimates for Doses Received During Normal Operation. For low doses and dose rates, a linear-quadratic model was found to provide a significantly better fit to the data for leukemia than a linear one, and leukemia risks were based on a linear-quadratic function. This reduces the effects by a factor of two over estimates that are obtained from the linear model. For other cancers, linear models were found to provide an adequate fit to the data, and were used for extrapolation to low doses. However, the BEIR V Committee recommended reducing these linear estimates by a factor between 2 and 10 for doses received at low dose rates. For this PEIS, a risk reduction factor of two was adopted for conservatism.

Table M.2.1.2–1. Lifetime Risks per 100,000 Persons Exposed to a Single Exposure of 10 rem

	Type of Fatal Cancer		
Gender	Leukemia[a]	Cancers Other Than Leukemia	Total Cancers
Male	220	660	880
Female	160	730	890
Average	**190**	**695**	**885**[b]

[a] These are the linear estimates, and are double the linear-quadratic estimates provided in BEIR V for leukemia at low doses and dose-rates.

[b] This value has been rounded up to 1,000 excess cancer fatalities per million person-rem.

Source: NAS 1990a.

Based on the above discussion, the resulting risk estimator would be equal to half the value observed for accident situations or approximately 500 excess fatal cancer per million person-rem (0.0005 excess fatal cancer per person-rem). This is the risk value used in this PEIS to calculate fatal cancers to the general public during normal operations. For workers, a value of 400 excess fatal cancers per million person-rem (0.0004 excess fatal cancer per person-rem) is used in this PEIS. This lower value reflects the absence of children (who are more radiosensitive than adults) in the workforce. Again, based on information provided in the *1990 Recommendations of the International Commission of Radiological Protection* (ICRP Publication 60), the health risk estimators for nonfatal cancer and genetic disorders among the public are 20 percent and 26 percent, respectively, of the fatal cancer risk estimator. For workers they are both 20 percent of the fatal cancer risk estimator. For this PEIS, only fatal cancers are presented.

The risk estimates may be applied to calculate the effects of exposing a population to radiation. For example, in a population of 100,000 people exposed only to natural background radiation (0.3 rem/yr), 15 latent cancer fatalities per year would be inferred to be caused by the radiation (100,000 persons x 0.3 rem/yr x 0.0005 latent cancer fatalities per person-rem = 15 latent cancer fatalities/yr).

Sometimes, calculations of the number of excess cancer fatalities associated with radiation exposure do not yield whole numbers and, especially in environmental applications, may yield numbers less than 1.0. For example, if a population of 100,000 were exposed as above, but to a total dose of only 0.001 rem, the collective dose would be 100 person-rem, and the corresponding estimated number of latent cancer fatalities would be 0.05 (100,000 persons x 0.001 rem x 0.0005 latent cancer fatalities/person-rem = 0.05 latent fatal cancers).

For latent cancer fatalities less than 1.0, the estimated 0.05 latent cancer fatalities is interpreted as a statistical estimate. That is, 0.05 is the *average* number of deaths that would result if the same exposure situation were applied to many different groups of 100,000 people. In most groups, no person (0 people) would incur a latent cancer fatality from the 0.001 rem dose each member would have received. In a small fraction of the groups, 1 latent fatal cancer would result; in exceptionally few groups, 2 or more latent fatal cancers would occur. The

average number of deaths over all the groups would be 0.05 latent fatal cancers (just as the average of 0, 0, 0, and 1 is 1/4, or 0.25). The most likely outcome is 0 latent cancer fatalities.

These same concepts apply to estimating the effects of radiation exposure on a single individual. Consider the effects, for example, of exposure to background radiation over a lifetime. The "number of latent cancer fatalities" corresponding to a single individual's exposure over a (presumed) 72-year lifetime to 0.3 rem/yr is the following:

1 person x 0.3 rem/year x 72 years x 0.0005 latent cancer fatalities/person-rem = 0.011 latent cancer fatalities.

Again, this should be interpreted in a statistical sense; that is, the estimated effect of background radiation exposure on the exposed individual would produce a 1.1-percent chance that the individual might incur a latent fatal cancer caused by the exposure over his full lifetime. Presented another way, this method estimates that approximately 1.1 percent of the population might die of cancers induced by the radiation background.

M.2.2 METHODOLOGY FOR ESTIMATING RADIOLOGICAL IMPACTS OF NORMAL OPERATION

The radiological impacts of normal operation of reactors and support facilities were calculated using Version 1.485 of the GENII computer code. Site-specific and technology-specific input data were used, including location, meteorology, population, food production and consumption, and source terms. [Text deleted.] Section M.2.2.1 briefly describes GENII and outlines the approach used for normal operations. The approach used for design basis accidents is discussed in Section M.5 of this appendix.

M.2.2.1 GENII Computer Code

The GENII computer model, developed by Pacific Northwest Laboratory for DOE, is an integrated system of various computer modules which analyze environmental contamination resulting from acute or chronic releases to, or initial contamination in, air, water, or soil. The model calculates radiation doses to individuals and populations. The GENII computer model is well documented for assumptions, technical approach, methodology, and quality assurance issues (GENII—The Hanford Environmental Radiation Dosimetry Software System, December 1988). The GENII computer model has gone through extensive quality assurance and quality control steps. These include the comparison of results from model computations against those from hand calculations, and the performance of internal and external peer reviews. Recommendations given in these reports were incorporated into the final GENII computer model, as deemed appropriate.

For this PEIS only the ENVIN, ENV, and DOSE computer modules were used. The codes are connected through data transfer files. The output of one code is stored in a file that can be used by the next code in the system. In addition, a computer code called CREGENII was prepared to aid and assist the user with the preparation of input files into GENII.

CREGENII. The CREGENII code helps the user, through a series of interactive menus and questions, to prepare a text input file for the environmental dosimetry programs. In addition, CREGENII prepares a batch processing file to manage the file handling needed to control the operations of subsequent codes and to prepare an output report.

ENVIN. The ENVIN module of the GENII code controls the reading of the input files prepared by CREGENII and organizes the input for optimal use in the environmental transport and exposure module, ENV. The ENVIN code interprets the basic input, reads the basic GENII data libraries and other optional input files, and organizes the input into sequential segments on the basis of radionuclide decay chains.

A standardized file that contains scenario, control, and inventory parameters is used as input to ENVIN. Radionuclide inventories can be entered as functions of releases to air or water, concentrations in basic environmental media (air, soil, or water), or concentrations in foods. If certain atmospheric dispersion options have been selected, this module can generate tables of atmospheric dispersion parameters that will be used in later calculations. If the finite plume air submersion option is requested in addition to the atmospheric dispersion calculations, preliminary energy-dependent finite plume dose factors also are prepared. The ENVIN module prepares the data transfer files that are used as input by the ENV module; ENVIN generates the first portion of the calculation documentation—the run input parameters report.

ENV. The ENV module calculates the environmental transfer, uptake, and human exposure to radionuclides that result from the chosen scenario for the user specified source term. The code reads the input files from ENVIN and then, for each radionuclide chain, sequentially performs the precalculations to establish the conditions at the start of the exposure scenario. Environmental concentrations of radionuclides are established at the beginning of the scenario by assuming decay of preexisting sources, considering biotic transport of existing subsurface contamination, and defining soil contamination from continuing atmospheric or irrigation depositions. Then, for each year of postulated exposure, the code estimates air, surface soil, deep soil, groundwater, and surface water concentrations of each radionuclide in the chain. Human exposures and intakes of each radionuclide are calculated for: 1) pathways of external exposure from finite atmospheric plumes; 2) inhalation; 3) external exposure from contaminated soil, sediments, and water; 4) external exposure from special geometries; and 5) internal exposures from consumption of terrestrial foods, aquatic foods, drinking water, animal products, and inadvertent intake of soil. The intermediate information on annual media concentrations and intake rates are written to data transfer files. Although these may be accessed directly, they are usually used as input to the DOSE module of GENII.

DOSE. The DOSE module reads the annual intake and exposure rates defined by the ENV module and converts the data to radiation dose. External dose is calculated with precalculated factors from the EXTDF module or from a data file prepared outside of GENII. Internal dose is calculated with precalculated factors from the INTDF module.

EXTDF. The EXTDF module calculates the external dose-rate factors for submersion in an infinite cloud of radioactive materials, immersion in contaminated water, and direct exposure to plane or slab sources of radionuclides. EXTDF was not used. Instead, the dose rate factors listed in *External Dose Rate Factors for Calculation of Dose to the Public* (DOE/EH-0070) were used for this PEIS.

INTDF. Using the *Limits for Intakes of Radionuclides by Workers* (ICRP Publication 30) model, the INTDF module calculates the internal (inhalation and ingestion) dose conversion factors of radionuclides for specific organs. The factors generated by INTDF were used for the calculations presented in this PEIS.

GENII is a general purpose computer code used to model dispersion, transport, and long-term exposure effects of specific radionuclides and pathways. Sophisticated codes such as UFOTRI and ETMOD (Environmental Tritium Model) are used exclusively for modelling tritium transport and dosimetry. The UFOTRI and ETMOD codes were not chosen for use in this PEIS due to the lack of information on detailed facility design and on the breakdown of tritium into elemental and tritiated water forms, and because these codes cannot be used for modeling the exposure effects of radionuclides other than tritium. GENII was chosen because it can model both air and surface transport pathways and is not restricted to any radionuclides.

M.2.2.2 Data and Assumptions

In order to perform the dose assessments for this PEIS, different types of data must be collected and/or generated. In addition, calculational assumptions have to be made. This section discusses the data collected and/or generated for use in the dose assessment and assumptions made for this PEIS.

Meteorological Data. The meteorological data used for all sites were in the form of joint frequency data files. A joint frequency data file is a table listing the fractions of time the wind blows in a certain direction, at a certain speed, and within a certain stability class. The joint frequency data files were based on measurements over a 1-year period at various locations and at different heights at the sites. Average meteorological conditions (averaged over the 1-year period) were used for normal operation. Meteorological data are presented in Health Risk Data, October 1996.

Population Data. Population distributions were based on *1990 Census of Population and Housing* data. Projections were determined for 2030 (approximate midlife of operations) for areas within 80 km (50 mi) of the proposed facilities at each candidate site. The site population in 2030 was assumed to be representative of the population over the operational period evaluated, and was used in the impact assessments. The population was spatially distributed on a circular grid with 16 directions and 10 radial distances up to 80 kilometers. The grid was centered on the facility from which the radionuclides were assumed to be released. Population data are presented in Health Risk Data, October 1996.

Source Term Data. The source terms (quantities of radionuclides released to the environment over a given period) were estimated on the basis of latest conceptual designs of facilities and experience with similar facilities. The source terms used to generate the estimated impacts of normal operation are provided in Section M.2.3 for the storage and disposition facilities which could be located at the various sites. Source terms for candidate and representative sites are presented in Sections M.2.4 through M.2.15.

Food Production and Consumption Data. Data from the *1987 Census of Agriculture* was used to generate site-specific data for food production. Food production was spatially distributed on the same circular grid as was used for the population distributions. The consumption rates were those used in GENII for the maximum individual and average individual. People living within the 80 km (50 mi) assessment area were assumed to consume only food grown in that area.

Calculational Assumptions. Dose assessments were performed for members of the general public and workers. Dose assessments for members of the public were performed for two different types of receptors considered in this PEIS: a maximally exposed offsite individual and the general population living within 80 km (50 mi) of the facility. It was assumed that the maximally exposed individual (MEI) was located at a position on the site boundary that would yield the highest impacts during normal operation of a given alternative. If more than one facility was assumed to be operating at a site, the dose to a "maximum receptor" (that is, a potential site MEI) from each facility was calculated. This was accomplished by preliminarily designating each potential MEI as a maximum receptor for each facility modeled—subsequently, whichever maximum receptor was found to incur the largest dose was then ultimately selected as the MEI for the site. An 80-km (50-mi) population dose was calculated for each operating facility at a site. These doses were then added to give the total population dose at that site.

To estimate the radiological impacts from normal operation of reactors and support facilities, additional assumptions and factors were considered in using GENII, as follows:

- No prior deposition of radionuclides on ground surfaces was assumed.
- For the maximally exposed off-site individual, the annual exposure time to the plume and to soil contamination was 0.7 year (NRC 1977b:1.109-68).
- For the population, the annual exposure time to the plume and to soil contamination was 0.5 year (NRC 1977b:1.109-68).
- The exposed individual or population was assumed to have the characteristics and habits (for example; inhalation and ingestion rates) of the adult human.

- A semi-infinite/finite plume model was used for air immersion doses. Other pathways evaluated were ground exposure, inhalation, ingestion of food crops and animal products contaminated by either deposition of radioactivity from the air or irrigation, ingestion of fish and other aquatic food raised in contaminated water, swimming and boating in contaminated surface water, and drinking contaminated water. It should be noted that not all pathways were available at every site.

- For atmospheric releases it was assumed that ground level releases would occur for all storage and disposal facilities. For site dependent facilities, reported release heights were used and assumed to be the effective stack height. Use of the effective stack height negates plume rise thereby making the resultant doses conservative.

- The calculated doses were 50-year committed doses from 1 year of intake.

The exposure, uptake, and usage parameters used in the GENII model are provided in Tables M.2.2.2–1 through M.2.2.2–4.

Annual average doses to workers for No Action at the Hanford Site (Hanford), Nevada Test Site (NTS), Idaho National Engineering Laboratory (INEL), Pantex Plant (Pantex), Rocky Flats Environmental Technology Site (RFETS), and Los Alamos National Laboratory (LANL) were generally based on measured values received by radiation workers during the 1989 to 1992 time period. The dose values are given in a series of documents that cover this time period. Dose values for 1992 are contained in "Compilation of Doses to Workers at DOE Facilities in 1992" (DOE 1993n:7). The same type reports are used for the earlier years. The average dose received by a worker at these sites in 2005 was assumed to remain the same as the annual average during the 1989 to 1992 period. The total workforce dose in 2005 was calculated by multiplying the average worker dose by the projected number of workers in 2005. For Oak Ridge Reservation (ORR) and Savannah River Site (SRS), worker dose projections provided by the sites were used. For NRC-licensed sites, No Action worker doses were based on reported values for 1993 given in *Occupational Radiation Exposure at Commercial Nuclear Power Reactors and Other Facilities, 1993* (NUREG-0713-V15).

Doses to workers directly associated with storage and disposition facilities were taken from the reports prepared by Fluor Daniel, Inc.; Sandia National Laboratories (SNL), New Mexico; LANL; Lawrence Livermore National Laboratory (LLNL); and SRS. To obtain the total workforce dose at a site with a particular storage or disposition facility in operation, the site dose from No Action was added to that from the storage or disposition facility being evaluated. The average dose to a site worker was then calculated by dividing this dose by the total number of radiation workers at the site.

All doses to workers include a component associated with the intake of radioactivity into the body and another component resulting from external exposure to direct radiation.

A more detailed discussion of doses to workers associated with storage and disposition is given in Section M.2.3.2.

M.2.2.3 Health Effects Calculations

In this PEIS, the collective combined effective dose equivalent is the sum of the collective committed effective dose equivalent (internal dose) and the collective effective dose equivalent (external dose), as explained in Section M.2.1.1. Doses calculated by GENII were used to estimate health effects using the risk estimators presented in Section M.2.1.2. The incremental cancer fatalities in the general population and groups of workers due to radiation exposure were therefore estimated by multiplying the collective combined effective dose equivalent by 0.0005 and 0.0004 fatal cancers/person-rem, respectively.Although health risk factors are statistical factors and therefore not strictly applicable to individuals, they have been used in the past to estimate the incremental risk to an individual from exposure to radiation. Therefore, the factor of 0.0005 and 0.0004

per rem of individual committed effective dose equivalent for a member of the public and for a worker, respectively, have also been used in this PEIS to calculate the individual's incremental fatal cancer risk from exposure to radiation.

For the public, the health effects expressed in this PEIS are the risk of fatal cancers to the maximally exposed individual and the number of fatal cancers to the 80-km (50-mi) population from exposure to radioactivity released from any site over the full operational period. For workers, the health effects expressed are the risk to the average worker at a site and the number of fatal cancers to all workers at that site over the full period of site operations.